BRIEF COMMUNICATION

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Portal Vein Thrombosis in Adult Omani Patients: A Retrospective Cohort Study

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ABSTRACT

Objectives: We sought to study the occurrence of portal vein thrombosis (PVT) in adult Omani patients. ***Methods***: We conducted a retrospective cross-sectional study in patients diagnosed with PVT, which was confirmed by radiological imaging, from two tertiary hospitals over a 10-year period. ***Results:*** Amongst the 39 patients enrolled in the study, 15 (38.4%) had cirrhosis of the liver, and 24 (61.5%) were non-cirrhotic. In the non-cirrhotic PVT patients, 15 (62.5%) had acute PVT, whereas nine (37.5%) had chronic PVT. PVT was more common in males than females, (25 (64.1%) vs. 14 (35.8%), respectively, $p = 0.020$). The three most common clinical symptoms were abdominal pain (n = 25, 64.1%) followed by nausea (n = 12, 30.7%) and fever (n = 8, 20.5%) patients. Causative risk factors included prothrombotic states (17.9–28.2%) and local factors (20.5%) such as cholecystitis, cholangitis, and liver abscess. Complications were found in 23.0% of patients with PVT, namely variceal bleeding in seven patients (17.9%) patients and bowel ischemia in two patients (5.1%). Management with sclerotherapy was performed in all patients with variceal bleeding. Thrombectomy was done for one patient complicated with intestinal ischemia, but as it failed, he was treated with warfarin anticoagulation. ***Conclusions:*** This is the first study reflecting a real-life practice in PVT with possibly underlying inherited and acquired prothrombotic conditions as well as complications due to local and malignant conditions from Oman. We studied the prevalence, clinical presentation, underlying possible etiological factors, treatment, and outcomes. Since causative factors were found in 36 patients (92.3%), etiological screening seems worthwhile in every case with PVT, but thrombophilia screening may not be cost-effective.

Portal vein thrombosis (PVT) refers to an obstruction in the trunk of the hepatic portal vein, originating from the confluence of the superior mesenteric and splenic veins posterior to the neck of the pancreas.[1,2] PVT is an important cause of portal hypertension (PH) and occurs either in association with cirrhosis or malignancy of liver, or rarely may occur without an associated liver disease.[3] Acute non-cirrhotic PVT, chronic non-cirrhotic PVT, and PVT with cirrhosis are the three main variants with varying etiological factors and presentation.[3,4] The incidence of PVT in Oman is currently unknown.

Generally, patients with acute PVT do not show features such as collateral circulation (e.g., cavernous portal transformation) or PH, as these are usually seen in chronic PVT. The terminology of extrahepatic portal venous obstruction (EHPVO) refers to the development of portal cavernoma in the absence of associated liver disease in chronic PVT.[3] The majority usually have a detectable underlying prothrombotic state.[5]

Accurate epidemiological data on PVT is difficult to obtain.[2] The prevalence of autopsy research in the Sweden, the United States, and Japan varied from 1%, 0.05% to 0.5% respectively.[5,6] However, of all the cases of PH in developing countries, 40% are attributed to PVT.[7] The incidence of PVT amongst patients with liver cirrhosis ranges from 0.6% to 64.1%, but EHPVO is the most common cause of PH globally.[2,6]

Anticoagulation is the mainstay of therapy for acute non-cirrhotic PVT. However, there is also some evidence to support its use in cirrhotic populations.[3,7] Chronic PVT (EHPVO), on the other hand, requires the management of PH as such with a role for anticoagulation in the setting of underlying prothrombotic state.[3,8] Thrombolysis via omental vein catheterization and the creation of transjugular intrahepatic portosystemic shunts

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(TIPS) and liver transplant may be feasible even in the setting of PVT. However, the proper selection of candidates and surgery type is warranted.[3,6–8] Thus, thrombolysis and thrombectomy also have a role.[6–8]

METHODS

This retrospective cohort study was conducted at two tertiary care hospitals in Oman (Royal Hospital and Armed Forces Hospital). The study was reviewed and approved by the Research and Ethics committees of the two centers. All patients admitted between January 2006 to June 2016 with a diagnosis of PVT were identified using the International Statistical Classification of Disease, version 10 (ICD-10) coding system in the hospital medical records. Only Omani patients, 16 years or older with a radiologically confirmed diagnosis of PVT using either ultrasound, computed tomography (CT) or magnetic resonance imaging (MRI) were included. The information of all patients fulfilling the criteria of PVT was entered in a data entry sheet. This included demographic data at the time of diagnosis, clinical features, comorbidities, laboratory and radiological investigations, and in-hospital management and outcomes [Table 1].

Basic investigations, such as complete blood count, serum electrolytes, renal and liver function tests, coagulation studies, blood sugar levels, and chest radiographs were recorded for all 39 patients. Thrombophilia screen included protein S, protein C, antithrombin, paroxysmal nocturnal hemoglobinuria (flow cytometry with CD55, CD59, and fluorescein-labeled proaerolysin (FLAER) expression), and JAK2 mutation. Antiphospholipid antibody screen included anti-cardiolipin, lupus anticoagulant, and anti-beta2-glycoprotein antibodies. In addition to the confirmatory diagnostic imaging, complication such as PH and variceal bleeding were diagnosed by endoscopy and bowel ischemia by CT. Importantly, we also noted the dates that these investigations were done and compared this to the date of PVT diagnosis to ascertain its relationship to PVT.

Patients were categorized as cirrhotic and non-cirrhotic types, with a further subclassification of the non-cirrhotic type into acute and chronic PVT, depending on the image of cavernoma transformation (formation of collateral blood vessels) or evidence of PH including splenomegaly and esophageal varices, as features of chronic PVT.

Table 1: Patient demographics, classification, presentation, risk factors, and complication in the portal vein thrombosis (PVT) patient cohort.

Patient characteristics	n (%)
Total	39 (100)
Male/female, n	25/14
Mean age, years	52.0
Range, years	21–81
Classification	
PVT in Cirrhosis	15/39 (38.4)
Non-cirrhotic PVT	24/39 (61.5)
Acute	15/24 (62.5)
Chronic	9/24 (37.5)
Presentation	
Abdominal pain	25 (64.1)
Nausea	12 (30.7)
Fever	8 (20.5)
GI bleeding	7 (17.9)
Weight loss	4 (10.2)
Ascites	11 (28.2)
Splenomegaly	15 (38.4)
Risk factors	
Acquired thrombophilic disorders,	7/39 (17.9)
[$]Myeloproliferative disorder	3/15 (20.0)
[#]Antiphospholipid	4/16 (25.0)
Inherited prothrombotic disorders,	11/39 (28.2)
Protein C deficiency	4/10 (40.0)
Protein S deficiency	5/10 (50.0)
Antithrombin deficiency	2/6(33.3)
Local factors,	8/39(20.5)
Cholecystitis	1 (2.5)
Cholangitis	6 (15.3)
Liver abscess	1 (2.5)
Malignancy,	13/39 (33.3)
Cirrhosis with HCC	8 (20.5)
Cholangiocarcinoma	3 (7.6)
Rectosigmoid carcinoma	1 (2.5)
Head of pancreas	1 (2.5)

[$] *JAK2.*
[#] *Anticardiolipid, lupus anticoagulant, anti-beta2-glycoprotein antibodies.*
GI: gastrointestinal; HCC: hepatocellular carcinoma.

Data was analyzed using SPSS Statistics (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.). Continuous data was expressed as mean and range. Qualitative data were expressed as percentages. The chi-square test was used to study the statistical significance of qualitative variables.

RESULTS

A total of 39 PVT patients were enrolled in this study; 15 (38.4%) showed cirrhosis of liver and 24 (61.5%) were classified as non-cirrhotic. Non-cirrhotic PVT patients were further subdivided as acute or chronic; 15 (62.5%) had an acute non-cirrhotic PVT, and nine (37.5%) had chronic PVT. PVT was more common in males than females (25 (64.1%) vs. 14 (35.8%), respectively, $p = 0.020$).

The presenting symptoms were abdominal pain (64.1%), nausea (30.7%), fever (20.5%), gastrointestinal bleeding (hematemesis or melena) (17.9%), and weight loss (10.2%). Demonstrable ascites and splenomegaly were seen in 28.2% and 38.5% of patients, respectively.

Amongst the 18 tested for an underlying thrombophilia, four (40.0%) had protein C deficiency, five (50.0%) had protein S deficiency, and two (33.3%) had antithrombin deficiency. Antiphospholipid antibodies (anti-cardiolipin, lupus anticoagulant, and anti-beta2-glycoprotein antibodies) were found in four patients (25.0%), and V617F JAK2 mutation was positive in three patients (20.0%). Factor V Leiden mutation, prothrombin G20210A mutation, hyperhomocysteinemia, and paroxysmal nocturnal hemoglobinuria (tested in 7, 4, 2, and 1 patients, respectively) were all negative.

The majority of non-cirrhotic PVT patients were tested for thrombophilia whereas only a few cirrhotic patients were tested. Furthermore, many patients were identified with more than one risk factor that may have contributed to the PVT. Local factors were seen eight patients (20.5%) with PVT; six patients had cholangitis, and one patient each had cholecystitis and liver abscess, respectively.

Cirrhosis of liver alone was the only risk factor in 15 patients (38.5%). However, eight patients (53.0%) with cirrhosis had hepatocellular carcinoma (HCC), which is a known risk factor for PVT. Thrombophilia is not routinely tested in cirrhotic patients; however, four patients with cirrhosis were tested for thrombophilia and were found to show protein C and S deficiency, which was expected due to an underlying acquired metabolic synthetic deficiency.

Thirteen patients (33.3%) in this study had an underlying malignancy. Eight (61.5%) cases with HCC all were found in PVT patients with cirrhosis. Additionally, in five patients (38.4%), other malignancies were observed within the acute non-cirrhotic PVT group. Three patients had cholangiocarcinoma, and one patient each had rectosigmoid carcinoma and carcinoma of the head of the pancreas. Furthermore, in three PVT patients (7.6%), no identifiable risk factors were seen.

Diagnosis was confirmed by CT in 29 patients (74.4%). In 12 patients (31.0%) the diagnosis was first established by an ultrasound and later confirmed by CT, but in six patients (15.3%) only an ultrasound was performed. In 12 PVT patients, MRI was done to look for underlying conditions (namely malignancy or hepatic parenchymal details). Four patients had undergone just an MRI, as the diagnosis was already established in other regional hospitals.

Endoscopy was done routinely in patients with cirrhosis as part of their assessment. However, in non-cirrhotic patients, it was done to rule out gastrointestinal bleeding in cases with a history of melena or hematemesis. The most common complication seen was PH in 10 (66.6%) PVT patients with cirrhosis in contrast to three (33.3%) chronic non-cirrhotic PVT patients.

Other complications in PVT patients with cirrhosis were portal hypertensive gastropathy (n = 7, 46.7%), esophageal varices (n = 7, 46.7%), gastric varices (n = 3, 20.0%), and variceal hemorrhage (n = 5, 33.3%). Chronic non-cirrhotic PVT patients had esophageal varices (n = 4, 44.4%), variceal hemorrhage (n = 2, 22.2%), and gastric varices (n = 1, 11.1%). Two (13.3%) patients with acute non-cirrhotic PVT presented with intestinal ischemia.

Eighteen (46.1%) patients with PVT received immediate anticoagulation with heparin followed by warfarin. Of these, 16 (88.8%) were non-cirrhotic and two (11.1%) had cirrhosis. Thrombectomy was done in one patient with PVT because it was complicated with intestinal ischemia; however, it failed. The patient was started on anticoagulation therapy with heparin followed by warfarin. Management with sclerotherapy was performed in all patients with variceal bleeding (17.9%). Transjugular portosystemic shunting was performed successfully in one patient. Patients who had PH (mainly patients with chronic PVT and cirrhosis) also received additional supporting treatment with beta-blocker prophylaxis and variceal banding.

Seven patients (46.6%) with acute non-cirrhotic PVT had a full recovery. Six (15.3%) patients died. Incidentally, 26 patients (66.6%) had no details on

follow-up as they were transferred to other local hospitals or were lost to follow-up.

DISCUSSION

Abdominal pain was the most common manifestation seen in almost two-thirds of the patient cohort and is explained by the fact that PVT is an intra-abdominal event and is thus most likely to present with abdominal symptoms. Kocher and Himmelmann reported a similar observation in their cohort of 20 non-cirrhotic PVT patients.[9] We observed that patients with non-cirrhotic PVT had more symptoms than cirrhotic PVT patients. Moreover, few non-cirrhotic PVT cases also presented with vomiting and watery diarrhea. Patients presenting with fever usually had intra-abdominal inflammatory processing like cholangitis. Expectedly, splenomegaly and ascites were seen more in cirrhotic patients with PVT. Detectable ascites by ultrasound was also seen in non-cirrhotic patients, which may be as a result or as a sequel of PH or bowel ischemia. Nevertheless, gastrointestinal bleeding (i.e., hematemesis or melena) was commonly seen with established PH and in chronic non-cirrhotic PVT.

Prothrombotic conditions, malignancy, and cirrhosis were the three main causative factors followed by local factors. Inherited prothrombotic disorders, acquired thrombophilic disorders, malignancy, local factors, and cirrhosis of the liver was 28.2%, 17.9%, 33.3%, 20.5%, and 38.4%, respectively. No identified cause was seen in three (7.6%) patients. This was in agreement with other studies of PVT in adults,[10,11] where cirrhosis was generally the most common cause followed by neoplasia. In many cases more than one known causative factors were identified and reported by others; however, the leading risk factor for PVT in our study was cirrhosis of liver (including HCC).[11,12]

Cirrhosis is now believed to be a hypercoagulable state, and its presentation may vary from being asymptomatic to having life-threatening issues.[8] Partial PVT is generally asymptomatic, whereas rapid, complete PVT presents typically with abdominal pain and non-bloody diarrhea.[8] Hypercoagulability in cirrhosis is multifactorial with increased factor VIII: C levels along with a reduction in albumin and protein C levels often tilting the balance.[13] Furthermore, bleeding risk appears to be higher in PVT patients with cirrhosis compared to patients with cirrhosis alone.[14]

Thrombophilia testing should not be performed during the period of acute thrombosis when these results can be falsely low. Further, these tests should be avoided if the patient is on active anticoagulation therapy. Thrombophilic conditions are usually identified in approximately 60% of patients and as an additional local predisposing factor in 30–40% of cases, with up to 80% cases demonstrating an underlying cause.[3] However, a meta-analysis on thrombophilia in PVT showed a prevalence of antithrombin deficiency (3.9%), protein C deficiency (5.6%), and protein S deficiency (2.6%), with odds ratios of 8.89, 17.63, and 8.00, respectively.[15] Myeloproliferative disorders (MPD) is also a common risk factor for PVT.[3] A meta-analysis showed that the mean prevalence of MPD and JAK2 mutation was 31.5% (95% CI: 25.1–38.8%) and 27.7% (95% CI: 20.8–35.8), respectively.[16] This compares well with the data from our cohort, showing that overall MPD with the JAK2 mutation was seen in 20% of patients (3 out of 15). Furthermore, in this cohort, protein C and protein S were tested in nine (23.0%) patients. It is our understanding that since most of our patients were on anticoagulation therapy, not all could be investigated for an underlying thrombophilia leading to the smaller number of patients being investigated. The role of hyperhomocysteinemia is still controversial. In this study, only two patients were tested, with both being found to be negative.

Local factors also play a causative role in PVT. In our study, the most common inflammatory local risk factors leading to PVT were cholangitis (2.5%), cholecystitis (15.3%), and liver abscess (2.5%), although a significant number of patients (33.3%) had malignancy including cholangiocarcinoma, rectosigmoid carcinoma, carcinoma of the head of pancreas, and HCC. In a Swedish study on 254 autopsies, 10% of patients with PVT had abdominal infections or inflammatory disease.[5] Furthermore, patients with cirrhosis and hepatic carcinoma had a high risk of PVT with an odds ratio of 17.1 (95% CI: 11.1–26.4).[5]

Although ultrasound is usually sufficient to make a diagnosis of PVT, CT is the tool of choice.[17,18] Furthermore, the sensitivity and specificity of MRI for detecting main PVT are 100% and 98%, respectively.[19] Moreover, both CT and MRI provide additional information such as extension

of thrombus, evidence of bowel infarction, and the status of adjacent organs. Thus, most cirrhosis patients are routinely followed-up with CT or ultrasound to look for complications or HCC, which would often be incidentally found in PVT as seen in our patient cohort.

PH was the most common complication in 13 patients (33.3%), followed by portal hypertensive gastropathy, esophageal, and gastric varices in agreement with other studies.[6–8]

It is therefore pertinent to have gastric endoscopy in patients with PVT as portal hypertensive gastropathy is more often present in the acute PVT, secondary to cancer or cirrhosis, whereas large esophageal varices are often present in patients with chronic PVT.[20] In chronic PVT, gastric varices were seen in up to 40% of patients.[21] In this cohort, seven patients (five patients with cirrhosis and two with chronic PVT) had evidence of variceal hemorrhage on endoscopy either at presentation or having developed 24 hours after admission. Two acute non-cirrhotic PVT patients presented with intestinal ischemia and were operated. One case was found to have developed cirrhosis related to chronic PVT.

Although complete recanalization is uncommon, complete or extensive recovery can be achieved with anticoagulant therapy.[3,22] Complete recanalization rates achieved with anticoagulation vary between 42–75% with the risk of extension of thrombus of only 5–7%.[3] Initial anticoagulation with heparin is generally followed-up with vitamin K antagonist like warfarin. However, with the recent availability of non-vitamin K antagonist like rivaroxaban, dabigatran, and apixaban, which have better efficacy and lower bleeding complications, the use of warfarin is expected to reduce.[23] Patients who received anticoagulation should be maintained on it for at least three months and continued thereafter depending on the underlying cause of thrombosis.

Treatment strategies most often include the use of anticoagulation, while thrombectomy and TIPS being considered as second-line options.[24,25] In this study, 46% of patients with PVT received anticoagulation: initially with low molecular weight heparin or heparin followed by warfarin, with one patient being shifted to rivaroxaban. The majority of these patients had non-cirrhotic PVT (87%) with only two patients (13%) having an underlying cirrhosis. One patient was treated with TIPS with no complication. This was in addition to other supportive standard treatments of PH and its complications which include the use of beta blockers, diuretics, and variceal banding. Overall, as it was shown by Kocher and Himmelmann,[9] the prognosis of acute non-cirrhotic PVT is good with complete recovery.

The study has many limitations. In particular, its small sample size, retrospective nature, and risk of biases. Although this study data collection spanned over a period of 10 years, we were able to obtain valid data from only 39 patients with PVT from the two institutions. Further, less than 50% of patients got tested for thrombophilia. This is actually a real-life scenario as thrombophilia testing cannot be performed initially during the phase of acute thrombosis (except for DNA studies), and in a majority of these patients who will get treated with anticoagulants, the window of opportunity to test for thrombophilia is only once the patients are off anticoagulants. However, a significant number of patients are lost to follow-up at this point in time. Lastly, one has to be careful in interpreting the thrombophilia testing results in this setting as well as remain alert to the fact that underlying liver cirrhosis will have some false positive results too.

CONCLUSIONS

This is the first study on PVT from Oman. We observed putative causative factors in 92.3% of the patient in the present cohort. Therefore, etiological screening is worthwhile in every case with PVT, but thrombophilia screening may not be cost-effective and has several logistic difficulties.

Disclosure
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